We claim:

1. A compound which specifically alters the binding activity of SR-BI, in combination with a pharmaceutically acceptable carrier, in an effective amount to treat a human or animal in need thereof, obtained by screening a library of compounds for alteration of SR-B1 binding activity or expression.

2. The compound of claim 1 selected from the group shown in Table I.

3. The compound of claim 1, selected from the group consisting of BLT-1 (MIT 9952-53), BLT-2 (MIT 9952-61), BLT-3 (MIT 9952-19), BLT-4 (MIT 9952-29), and BLT-5 (MIT 9952-6).

4. A method for altering cholesterol transport into or out of cells comprising inhibiting expression or activity of SR-BI comprising administering to an animal or human in need thereof the composition of claim 1.

5. The method of claim 4, wherein the composition of claim1 enhances HDL binding by increasing SR-BI's binding affinity for HDL.

6. The method of claim 4, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated lipid transport.

7. The method of claim 6, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated selective lipid uptake.

8. The method of claim 7, wherein the lipid is HDL cholesteryl ether.

9. The method of claim 4, wherein the inhibited SR-BI binding activity blocks efflux of cellular cholesterol to HDL.

10. A method of identifying a compound which alters SR-BI binding activity or expression comprising screening a library of compounds.

11. The method of claim 10, wherein the SR-BI expression is determined by Northern analysis.

12. The method of claim 10, wherein the library is a chemical library.

13. The method of claim 10, wherein the SR-BI binding activity is inhibited.

14. The method of claim 13, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated lipid transport.

15. The method of claim 14, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated selective lipid uptake.

16. The method of claim 15, wherein the lipid is HDL cholesteryl ether.

17. The method of claim 10, wherein the inhibited SR-BI binding activity blocks efflux of cellular cholesterol to HDL.